

Bleeding Gastric Varices: Frequency and Outcome

Khairy H. Morsy^{1*}, Asmaa N. Mohammad¹, Mahmoud K. El-Samman², Walaa A. Ali

¹Department of Tropical Medicine and Gastroenterology, Faculty of Medicine, Sohag University, Sohag, Egypt

²Department of Internal Medicine, Faculty of Medicine, Sohag University, Sohag, Egypt

***Corresponding Author:** Khairy H. Morsy, Department of Tropical Medicine and Gastroenterology, Faculty of Medicine, Sohag University, Sohag, Egypt, **Email:** khairy.morsy@yahoo.com

Abstract

Background and aims: Gastric variceal bleeding is less frequent than esophageal varices bleeding but it still a serious cause of morbidity and mortality. The aim of study was to assess the frequency and identify the patients' outcome after management.

Patients and methods: The study was conducted on cirrhotic patients with upper gastrointestinal bleeding in the period from June 2016 to June 2017 who underwent esophago-gastro-duodenoscopy & subjected to complete history taking, clinical examination, laboratory investigations & abdominal US.

Results: The study included 500 patients. In the endoscopic findings, bleeding from gastric varices (GV) was seen in 50 patients (10 %), bleeding from esophageal (EV) was detected in 400 patients (80 %), while bleeding from other sources was in 50 patients (10%). The mean age of GV patients was 56.64± 11.06; most of them were males 34 (68 %) & the most common etiology of the underlying liver cirrhosis & portal hypertension was HCV that was detected in 40 patients (80%). Only 35 patients (70%) had isolated GV type I & 15 patients (30%) had continuous Gastroesophageal varices type 2 with red colour signs (Rc+) in 80% of the cases. PHG was seen in 48 patients (96%). Bleeding was more common between Child A patients although it was not significant. Also there were no significant differences between grades of gastric varices according to MELD score. As regards the outcome, 40% developed eradication, 38% died & 22% developed re-bleeding. Upon studying the predictors of mortality, we found that they had significantly lower albumin & higher ALT & AST levels. Early re-bleeding was more common among child A patients with moderately sized GV (F2), but the difference was not statistically significant. By multivariate analysis we found that there were no independent predictors of rebleeding gastric varices.

Conclusion: frequency of bleeding GV was 10% among cirrhotic patient with upper GIT bleeding. No independent factors could predict the mortality or rebleeding among cirrhotic patients with bleeding GV by multivariate analysis.

Keywords: Gastric varices, Esophageal varices, Bleeding.

1. INTRODUCTION

Acute upper gastrointestinal (GI) bleeding is a common cause of emergency hospitalization worldwide¹. Acute variceal hemorrhage (AVH) is an important complication of portal hypertension (PH) that is associated with significant morbidity and mortality². Gastric variceal bleeding is a serious cause of morbidity and mortality among patients with PH. Most of the studies underestimate their true prevalence³. It may be difficult to distinguish fundal varices from gastric folds due to their deep submucosal location as well as the normal color and appearance of the overlying mucosa⁴. The prevalence of gastric varices (GV) in patients with PH varies from 18 to 70%; although the incidence of bleeding from GV is relatively low ranging from 10 to 36%. Management of GV

presents a challenging problem, because since there is no consensus regarding the optimum treatment of GV, treatment tends to be empirical⁵. Sarin et al⁶, documented that GV were found in 20% of patients with PH and secondary GV developed in 9% of patients during follow-up evaluation.

Although GV bleeding occurs less frequently than esophageal varices (EV) bleeding; it tends to be more severe and requires more blood transfusions with a higher mortality than EV bleeding⁶. Red color spots, larger nodular gastric varices, and fundal location have been identified as risk factors for GV bleeding⁷. In addition, Kim et al⁸. Found that advanced Child-Pugh class, varix > 5 mm in size, and the presence of a red spot were associated with an increased risk for a first bleed. As a result of the

high morbidity and mortality of GV, it is valuable to estimate its exact frequency and identify risk factors for bleeding among upper Egyptian patients. In addition, evaluation of GV outcome after GV injection.

2. AIM OF THE WORK

The aim of study was to assess frequency of bleeding GV and identify the patients' outcome after management.

3. PATIENTS & METHODS

This prospective clinical study was conducted on all cirrhotic patients with upper GIT bleeding seeking medical advice at the Tropical Medicine and Gastroenterology Department, Sohag University Hospital in the period from June 2016 to June 2017.

3.1. Inclusion Criteria

Patients with liver cirrhosis and portal hypertension underwent EGD after upper GIT bleeding at the Tropical Medicine and Gastroenterology Department, Sohag University Hospital and diagnosed to have bleeding gastric varices.

3.2. Exclusion criteria

Patients diagnosed to have other causes of upper GIT bleeding (such as: EV, peptic ulcer disease, reflux esophagitis, erosions, antral vascular ectasia).

All patients will be subjected to the following:

1- Complete clinical evaluation.

2- Laboratory Investigation: Complete blood picture, Liver profile serological testing for HBV, and HCV, Serum Creatinine, Ascitic fluid study.

3- Abdominal Ultrasonography :With details about Size of the liver, surface, hepatic focal lesion, portal vein diameter, portal vein thrombosis, Spleen size, & porto-systemic collaterals and ascites.

4- Upper GIT endoscopy: Upper endoscopic examination for detailed evaluation of the esophagus, stomach and duodenum.

Table1. Frequency of bleeding gastric varices among cirrhotic patients with upper gastrointestinal bleeding

Causes of bleeding	Number (N=50)(Percentage)
Gastric varices	50 (10%)
Esophageal varices	400 (80%)
Gastric ulcers	30 (6%)
Portal hypertensive gastropathy	20 (4%)

4. ETHICAL CONSIDERATION

The study design was approved by the ethical and scientific research committee of Faculty of Medicine, Sohag University. Informed consents were obtained from all patients.

5. RESULTS

In the endoscopic findings, bleeding from gastric varices was seen in 50 patients (10 %) , bleeding from esophageal varices was detected in 400 patients (80 %), while bleeding from other sources (non variceal _ bleeding) was in 20 patients (4%) as shown in Table (1) .The mean age of the studied population was 56.64± 11.06, most of them were males 34 (68 %) Table (2).The most common etiology for the liver cirrhosis & portal hypertension of the studied group was HCV which was detected in 40 patients (80%) While HBV was detected in 2 patients only (4%) as shown in Table (2). Primary hemostasis was achieved in all patients with histoacryl injection. Only 35 patients (70%) had isolated GV type I & 15 patients (30%) had continuous Gastroesophageal varices type 2 with red colour signs (Rc+) in 80% of the cases. Bleeding from OVS was in 38 patients (76%) & thoses who underwent band ligation were 31 patients (62%).PHG was seen in 48 patients (96%) as shown in Table (4). Bleeding was more common between Child A patients but there were no significant differences as regard child classification or size of varices, also MELD score as shown in Tables (3), (5). Rate of rebleeding was (22%) , deaths occurred in 19 patients (38%) after admission & eradication of GVs were achieved in 20 (40%)Table(6).Upon studying the predictors of mortality, we found that they had significantly lower albumin & higher ALT & AST levels with no significant difference as regard ultrasonographic findings Table (7). Regarding to child classification & MELD score we found no significant difference between the two groups and these scores could not predict the mortality in our Patients Table (8).By multivariate analysis we found no independent predictors of mortality or rebleeding in cirrhotic patients with gastric varices as shown in Table (9) & (12).

Table2. Demographic, clinical, laboratory & ultrasonographic data of the studied group

Variable	Summary statistics
Age/years Mean ± SD	56.64±11.06
Gender Female/Male	16 (32.00%)/ 34 (68.00%)
Smoking	24 (48.00%)
Beta - Blockers	31 (62.00%)
Non-steroidal anti-inflammatory drugs	5 (10.00%)
Diabetes mellitus	6 (12.00%)
Hypertension	1 (2.00%)
Number of attacks	
First	17 (34.00%)
Second	15 (30.00%)
Third	10 (20.00%)
Fourth	5 (10.00%)
Fifth	2 (4.00%)
Sixth	1 (2.00%)
H/O of Portal hypertension	32 (64.00%)
Bilharziasis	6 (12.00%)
pulse rate Mean ± SD	82.72±9.75
Systolic blood pressure Mean ± SD	108.2±13.04
Diastolic blood pressure Mean ± SD	70.1±8.83
Pallor	20 (40.00%)
Jaundice	7 (14.00%)
Mental status Conscious /Disturbed	47 (94.00%) /3 (6.00%)
Flapping	15 (30.00%)
Lower limb edema	16 (32.00%)
Hepatomegaly	Zero
Splenomegaly	10 (20.00%)
Ascites	10 (20.00%)

Table3. Child classification and MELD score among studied population

Variable	Summary statistics
Child score A/B/C	32 (64.00%)/9 (18.00%)/9 (18.00%)
MELD score Mean ± SD	11.63±6.57

Table4. Upper endoscopy findings in studied populations

Variable	Summary statistics
OVS	38 (76.00%)
EVL	31 (62.00%)
PHG	48 (96.00%)
Fundal varices F1/F2/ F3	3 (6.00%)/37 (74.00%)/10 (20.00%)
Red colour signs	40 (80 %)
Histoacryl Injection	50 (100%)
Previous Histoacryl Injection	19 (38.00%)
Types of fundal varices	
Isolated (type I)/Continuous (type 2)	35 (70 %)/ 15 (30 %)

Table5. Relation between Fundal varices& Child and MELD scores in studied population

Variable	F1(N=3)	F2(N=37)	F3(N=10)	P value
Child score				
A	1 (33.33%)	24 (64.86%)	7 (70.00%)	0.78
B	1 (33.33%)	6 (16.22%)	2 (20.00%)	
C	1 (33.33%)	7 (18.92%)	1 (10.00%)	
MELD score Mean ± SD	16.4±9.81	11.36±6.44	11.21±6.30	0.63

Table6. Outcome of studied population

Variable	Summary statistics
Eradication	20 (40.00%)
Re-bleeding	11 (22.00%)
Death	19 (38.00 %)

Table 7. Comparison between survivors & non-survivors as regard demographic, clinical, laboratory & ultrasonographic data

Variable	Survivors (31)	Death(19)	P value
Age/years Mean \pm SD	55.10 \pm 12.29	59.16 \pm 8.38	0.21
Gender Female	9 (29.03%)	7 (36.84%)	0.57
male	22 (70.97%)	12 (63.16%)	
Smoking	16 (51.61%)	8 (42.11%)	0.51
Beta Blockers	19 (61.29%)	12 (63.16%)	0.90
Non-steroidal anti- inflammatory drugs	3 (6.68%)	2 (10.53%)	1.00
Diabetes mellitus	4 (12.90%)	2 (10.53%)	1.00
Hypertension	1 (3.23%)	0	1.00
Number of attacks			0.60
First	11 (35.48%)	6 (31.58%)	
Second	8 (25.81%)	7 (36.84%)	
Third	7 (22.58%)	3 (15.79%)	
Fourth	3 (9.68%)	2 (10.53%)	
Fifth	2 (6.45%)	0	
Sixth	0	1 (5.26%)	
H/O of Portal hypertension	18 (58.06%)	14 (73.68%)	0.26
Bilharziasis	3 (6.68%)	3 (15.79%)	0.66
Chronic Hepatitis	17 (54.84%)	11 (57.89%)	0.83
pulse rate Mean \pm SD	83.10 \pm 10.40	82.11 \pm 8.81	0.73
Systolic blood pressure Mean \pm SD	109.03 \pm 13.00	106.84 \pm 13.35	0.57
Diastolic blood pressure Mean \pm SD	70.65 \pm 8.92	69.21 \pm 8.86	0.58
Pallor	12 (38.71%)	8 (42.11%)	0.81
Jaundice	4 (12.90%)	3 (15.79%)	1.00
Mental status			1.00
Conscious	29 (93.55%)	18 (94.74%)	
Disturbed	2 (6.45%)	1 (5.26%)	
Flapping	7 (22.58%)	8 (42.11%)	0.14
Lower limb edema	8 (25.81%)	8 (42.11%)	0.23
Splenomegaly	6 (19.35%)	4 (21.05%)	1.00
Ascites	6 (19.35%)	4 (21.05%)	1.00
haemoglobin (gm/dl) Mean \pm SD	8.74 \pm 2.03	8.65 \pm 1.66	0.88
Mean corpusler volume Mean \pm SD	82.23 \pm 10.67	83.31 \pm 10.41	0.73
White blood cells (k/ul) Mean \pm SD	8.76 \pm 5.17	9.22 \pm 3.51	0.42
Platelets (k/ul) Mean \pm SD	124.26 \pm 39.31	116.47 \pm 41.58	0.51
Bilirubin (mg/dl) Mean \pm SD	1.50 \pm 1.27	2.52 \pm 2.10	0.049
Albumin (gm/dl) Mean \pm SD	3.41 \pm 0.59	2.94 \pm 0.73	0.02
Prothrombine time (seconds) Mean \pm SD	16.14 \pm 2.27	16.96 \pm 2.77	0.26
Prothrombine concentration Mean \pm SD	66.40 \pm 13.10	63.63 \pm 13.89	0.48
International normalization rate Mean \pm SD	1.25 \pm 0.31	1.41 \pm 0.31	0.08
Alanine transaminase (u/l) Mean \pm SD	77.06 \pm 172.82	85.42 \pm 80.89	0.01
Aspartate transaminase (u/l) Mean \pm S	108.71 \pm 285.84	115.79 \pm 140.72	0.04
Hepatitis B virus	2 (6.45%)	0	0.52
Hepatitis C virus	22 (70.97%)	18 (94.74%)	0.07

Creatinine (mg/dl) Mean ± SD	0.94±0.42	1.74±2.09	0.08
Potassium (mmol/l) Mean ± SD	3.65±0.65	3.73±1.24	0.77
Hepatic focal lesion	1 (3.23%)	4 (21.05%)	0.06
Portal vein Not dilated Dilated	7 (22.58%) 24 (77.42%)	5 (26.32%) 14 (73.68%)	0.76
Portal vein thrombosis	4 (12.90%)	5 (26.32%)	0.23
Splenomegaly	28 (90.32%)	17 (89.47%)	1.00
Ascites	11 (35.48%)	6 (31.58%)	0.78

Table8. Comparison between survivors & non survivors as regard child classification and MELD score

Variable	Survivors (N=31)	Non Survivors (N=19)	P value
Child score			
A	23 (74.19%)	9 (47.37%)	0.16
B	4 (12.90%)	5 (26.32%)	
C	4 (12.90%)	5 (26.32%)	
MELD score Mean ± SD	11.71±7.16	11.5±5.67	0.83

Table9. Multivariate analysis for predictors of mortality

Variable	Odds ratio (95% confidence interval)	P value
Bilirubin	1.11 (0.64-1.88)	0.72
Albumin	0.67 (0.16-2.74)	0.58
INR	2.00 (0.19-20.84)	0.56
ALT	1.01 (0.97-1.05)	0.54
AST	0.99 (0.97-1.02)	0.51
HCV vs. No HCV	3.18 (0.40-36.24)	0.24
Creatinine	1.45 (0.53-4.00)	0.46
HFL vs. HFL	3.18 (0.19-53.53)	0.42

Table10. Univariate analysis for predictors of rebleeding (Demographic and clinical data).

Variable	No re-bleeding	Re-bleeding	P value
Age/years Mean ± SD	56.79±12.47	56.17±4.60	0.87
Gender			
Female	12 (31.58%)	4 (33.33%)	0.91
Male	26 (68.42%)	8 (66.67%)	
Smoking	18 (50.00%)	5 (41.67%)	0.61
B. Blockers	24 (63.16%)	7 (58.33%)	0.76
NSAID	3 (7.89%)	2 (16.67%)	0.58
DM	3 (7.89%)	3 (25.00%)	0.14
Hypertension	1 (2.63%)	0	1.00
Number of attacks			
First	12 (31.58%)	5 (41.67%)	0.92
Second	11 (28.95%)	4 (33.33%)	
Third	8 (21.05%)	2 (16.67%)	
Fourth	4 (10.53%)	1 (8.33%)	
Fifth	2 (5.26%)	0	
Sixth	1 (2.63%)	0	
H/O of portal hypertension	27 (71.05%)	5 (41.67%)	0.09
Bilharziasis	6 (15.79%)	0	0.31
Hepatitis	20 (52.63%)	8 (66.67%)	0.39
Pulse rate Mean ± SD	81.63±10.19	86.17±7.50	0.16
Systolic blood pressure Mean ± SD	107.11±11.83	111.67±16.42	0.30
Diastolic blood pressure Mean ± SD	69.34±8.56	72.5±9.65	0.29
Pallor	13 (34.21%)	7 (58.33%)	0.18
Jaundice	6 (15.79%)	1 (8.33%)	1.00

Mental status			
Conscious	36 (94.74%)	11 (91.67%)	1.00
Disturbed	2 (5.26%)	1 (8.33%)	
Flapping	11 (28.95%)	4 (33.33%)	1.00
Lower limb edema	14 (36.84%)	2 (16.67%)	0.29
Splenomegaly	8 (21.05%)	2 (16.67%)	1.00
Ascites	9 (23.68%)	1 (8.33%)	0.42

Table11. Univariate analysis for predictors of rebleeding(Lab. And US)

Hb Mean ± SD	8.89±1.98	8.12±1.45	0.22
MCV Mean ± SD	82.64±10.94	82.62±9.30	0.99
WBCs Mean ± SD	9.17±3.78	8.21±6.66	0.12
PLTs Mean ± SD	115.87±38.14	138.5±42.32	0.09
Bilirubin Mean ± SD	2.15±1.83	1.07±0.60	0.051
Albumin Mean ± SD	3.16±0.71	3.45±0.50	0.20
PT Mean ± SD	16.59±2.62	16.03±1.99	0.51
PC Mean ± SD	65.55±13.92	64.73±11.80	0.85
INR Mean ± SD	1.34±0.32	1.23±0.29	0.31
ALT Mean ± SD	67.79±61.08	119.67±278.31	0.07
AST Mean ± SD	87.21±103.57	188±459.11	0.31
HBV	1 (2.63%)	1 (8.33%)	0.43
HCV	32 (84.21%)	8 (66.67%)	0.23
Creatinine Mean ± SD	1.34±1.55	0.93±0.38	0.58
K Mean ± SD	3.78±0.96	3.38±0.69	0.20
Cirrhosis	38 (100%)	12 (100%)	
HFL	5 (13.16%)	0	0.32
PV	30 (78.95%)	8 (66.67%)	0.45
PVT	8 (21.05%)	1 (8.33%)	0.43
Splenomegaly	35 (92.11%)	10 (83.33%)	0.58
Ascites	13 (34.21%)	4 (33.33%)	1.00

Table12. Multivariate analysis of re-bleeding predictors

Variable	Odds ratio (95% confidence interval)	P value
PLTs	1.00 (0.98-1.03)	0.70
Bilirubin	0.30 (0.07-1.33)	0.11
ALT	1.00 (0.99-1.01)	0.24
MELD score	0.95 (0.77-1.19)	0.68

6. DISCUSSION

In our present study, frequency of bleeding gastric varices was 10 %, frequency of esophageal bleeding was 80% while non-variceal bleeding (gastric ulcers & PHG) represented only 10% .Similar studies reported that gastric varices (GV) are responsible for 10-30% of all variceal hemorrhage⁹.

In another study, Gastric varices are responsible for about 10% of variceal bleeding; however, they are also the cause of massive haemorrhage, often with dramatic progress¹⁰ .Recently, Mumtaz and his collages (in Pakistan) reported that the overall prevalence of gastric varices among their patients was 15% ¹¹.Another prospective observational study in which 589 UGIB patients underwent upper gastrointestinal endoscopy from May 2010 to April 2013 in Nepal. Among UGIB patients, 33.1 % were variceal and 66.9 % were non-variceal bleeding (peptic ulcers 23.9 %, gastric erosion 16.5 %

and others)¹². The mean age of the studied population was 56.64± 11.06 year, most of them were males 34 (68 %). In another study, out of 205 patients undergoing esophago-gastroduodenoscopy, 122(59.5%) were male and 83(40.5%) were female with an overall mean age of 49.5+/-7.49 years¹³.While a total of 41 patients (39 with cirrhosis) underwent 54 cyanoacrylate injections during study period. Mean age was 57 and 73 % were males¹⁴.It was found by our study that the most common etiology of portal hypertension which led to GV bleeding was liver cirrhosis (100%).Another observational cross-sectional study was conducted from August 2014 to August 2015 at Aziz Bhatti Shaheed Teaching Hospital, Gujrat, Pakistan, using non-probability consecutive sampling. Patients having liver cirrhosis only due to chronic hepatitis C virus, portal vein diameter >12mm or spleen size >12cm in long axis and ascites with no previous history of variceal banding were included. Out of 205

patients undergoing esophago-gastroduodenoscopy, 122(59.5%) were male and 83(40.5%) were female with an overall mean age of 49.5+/-7.49 years. Gastric varices were present in 30(14.6%) patients¹³ and between January 1998 and January 2010, 97 patients with gastric variceal bleeding underwent endoscopic treatment with a mixture of N-butyl-2-cyanoacrylate and Lipiodol, ninety-one patients had cirrhosis and 6 had non-cirrhotic portal hypertension¹⁵. The medical records of patients who presented with active gastric variceal bleeding between 1998 and 2011 in a tertiary care setting were evaluated retrospectively and the eventual outcome(s) (initial hemostasis, rebleeding, and mortality rate) was assessed at least 1 year after the index bleed (ninety-seven patients were included), the mean age of the patients was 51.0 +/- 12.5 years; 62% were men.

Hepatitis C was the most common etiology, found in 63 (65%) patients¹⁶. Our present study showed that the population study who underwent EGD had EV (76%), moderately filled bleeding GVs (74%). Isolated gastric varices type 1 & gastroesophageal varices type 2 had higher bleeding incidence (70%, 30%) respectively. Similar results were reported by Sarin et al.,⁶ where isolated gastric varices type 1 and gastroesophageal varices type 2 had a significantly higher bleeding incidence (78% and 55% respectively), than gastroesophageal varices type 1 and isolated gastric varices type 2 (10%). However, Mumtaz et al.,¹¹ reported that the incidence of bleeding from isolated gastric varices type 1 was 36% and from gastroesophageal varices type 1, gastroesophageal varices type 2 and isolated gastric varices type 2 were 17.7%, 20.5% and 0% respectively. Butt et al.,¹³ show that gastric varices were present in minority of patients undergoing esophago-gastroduodenoscopy, and among them gastroesophageal varices type 1 was the most common, while isolated gastric varices type 2 was not present in any patient. Other similar results from Egypt were reported by Thakeb and his college, where isolated gastric varices were accounting for 7.2% and gastroesophageal varices were accounting for 27.1%¹⁷. In our study, there was also a relation between bleeding from isolated gastric varices and endoscopic signs where bleeding was more in patients with red color signs as reported in 80% of the cases. The same results were reported by others where they found a significant relation between bleeding from gastric varices and the

presence of red color signs¹⁸. However, there was non statistically significant relation between bleeding gastric varices, MELD & Child scores where bleeding was more in patients with Child class A. This was attributed to small number of the studied population (50 patients) & also limited duration of the study through which data were obtained. This was against other studies, such as the results which were reported by Kim et al., 1997⁸ where bleeding from gastric varices was more common in patients with Child class B and C (80% of bleeder) than in patients with Child class A (20% of bleeders) and also that difference was statistically significant. Another author reported Child-Pugh score at presentation for cirrhotic patients was A-12.1%, B-53.8%; C-34.1% and median MELD score at admission was 13 (3-26)¹⁵. Child-Pugh classification was a significant prognostic factor of survival ($P < 0.001$)¹⁶. While other reports failed to find a significant relation between bleeding from gastric varices and the severity of liver dysfunction¹¹. There were no definite risk factors for bleeding GV as our study didn't have control group to identify them by comparison with the study group, while other studies had. The presence of (ulcers and red color sings) and severe liver dysfunction (Child class B and C) were found to be independent risk factors for bleeding from GV regardless their type, there are many studies reported the same risk factors for bleeding from gastric varices but with minimal differences in between them⁸. In our present study, 40% of the studied population developed eradication 38% died & 22% developed re-bleeding. Histoacryl injection achieved Successful primary hemostasis in all patients with bleeding gastric varices and early rebleeding occurred in only 11 cases (24%).

Secondary hemostasis was achieved in all of them using N-butyl 2 cyanoacrylate (Histoacryl) with no serious complications have been reported. Mumtaz et al., 2007¹¹ reported that primary hemostasis was achieved with histoacryl injection in 100% of patients and early rebleeding rate was 14%. Mortality was less than our study in others such there were 132 patients with acute GVB (83.3% men, median age 51.3 years) with endoscopic NBC injection treatments recruited. Mortality within 6 weeks was noted in 16.7% patients¹⁹. In another study, a total of 20 patients (15 male) with PH and GV were enrolled and treated with undiluted NBC injection. The patients were followed clinically and endoscopically for a median of 31 months. Eradication of GV was observed in all patients

(13 patients were treated with 1 session and 7 patients with 2 sessions). Overall, GV recurrence was observed in 2 patients (10%), after 6 and 35 mo of follow-up, and GV bleeding rate was 5% (1 patient)²⁰. Upon studying the predictors of mortality, we found that they had significantly lower albumin & higher ALT & AST levels with no significant difference as regard ultrasonographic & endoscopic findings. Regarding to child classification & MELD score we found no significant difference between the two groups and these scores could not predict the mortality in our patients. Impaired renal function, deteriorated liver function with Child pugh score ≥ 9 as well as MELD score ≥ 18 , rebleeding within 5 days, and acute on top liver cell failure are independent predictors of mortality¹⁹. We found in our study that early re-bleeding was more common among child A patients with moderately sized GV (F2), but the difference was not statically significant & by multivariate analysis we found that were no independent predictors of rebleeding gastric varices.

Other studies reported that predictors for rebleeding are usually related to the severity of the bleeding and characteristics of the ulcer, whereas advanced age, physical status of the patient, and comorbidities are important predictors for mortality in addition to those for rebleeding²¹. Patients with AVH and MELD score ≥ 18 , requiring ≥ 4 units of PRBCs within the first 24 h or with active bleeding at endoscopy are at increased risk of dying within 6 weeks. MELD score ≥ 18 is also a strong predictor of variceal re-bleeding within the first 5 days²².

Another study reported that re-bleeding within 5 days occurred in 37 patients (15%), MELD score ($p = 0.01$) and a clot on a varix ($p = 0.05$) predicted re-bleeding. Patients with a MELD score ≥ 18 ; both MELD score ≥ 18 and ≥ 4 units of PRBCs transfused; both MELD score ≥ 18 and active bleeding at index endoscopy; and variceal re-bleeding had increased risk of death 6 weeks post-AVH²².

7. CONCLUSION

No independent factors could predict the mortality or rebleeding among cirrhotic patients with bleeding GV by multivariate analysis.

REFERENCES

- [1] Kapsoritakis AN, Ntounas EA, Makrigiannis EA, Ntouna EA, Lotis VD, Psychos AK, Et al: Acute upper gastrointestinal bleeding in central Greece: the role of clinical and endoscopic variables in bleeding outcome. *Digestive diseases and sciences*. 54(2):333-41(2009) .
- [2] Carbonell N, Pauwels A, Serfaty L, Fourdan O, Levy VG, Poupon R. Improved survival after variceal bleeding in patients with cirrhosis over the past two decades. *Hepatology* (Baltimore, Md). 40(3):652-9 (2004) .
- [3] BM Ryan, RW Stockbrugger, JM Ryan – Gastroenterology. Pathophysiologic , gastroenterologic and radiologic approach to mangement of gastric varices. 126(4) : 1175-1189.(2004)
- [4] Sanyal AJ. The value of EUS in the management of portal hypertension. *Gastrointest Endosc*. 52:575–7 (2000) .
- [5] Tajiri T, Onda M, Yoshida H, Mamada Y, Tani N, Yamashita K. The natural history of gastric varices. *Hepatogastroenterology*. 49: 1180-2 (2002).
- [6] Sarin, S. K. & Lahoti, D. Management of gastric varices. *Baillieres Clin Gastroenterol*, 6, 527-48(1992) .
- [7] Hashizume, M., Kitano, S., Yamaga, H., Koyanagi, N. & Sugimachi, K. Endoscopic classification of gastric varices .*Gastrointest Endosc*, 36, 276-80 (1990) .
- [8] Kim, T., Shijo, H., Kokawa, H., Tokumitsu, H., Kubara, K., Ota, K., Akiyoshi, N., Iida, T., Yokoyama, M. & Okumura, M. Risk factors for hemorrhage from gastric fundal varices. *Hepatology*, 25, 307-12 (1997) .
- [9] Wani, Z. A., Bhat, R. A., Bhadoria, A. S., Maiwall, R. & Choudhury, A Gastric varices: Classification, endoscopic and ultrasonographic management. *J Res Med Sci*, 20, 1200-7(2015).
- [10] Koziel, S., Kobryn, K., Paluszkiwicz, R., Krawczyk, M. & Wroblewski, T. Endoscopic treatment of gastric varices bleeding with the use of n-butyl-2 cyanoacrylate. *Prz Gastroenterol*, 10, 239-43 (2015) .
- [11] Mumtaz, K., Majid, S., Shah, H., Hameed, K., Ahmed, A., Hamid, S. & Jafri, W. Prevalence of gastric varices and results of sclerotherapy with N-butyl 2 cyanoacrylate for controlling acute gastric variceal bleeding. *World J Gastroenterol*, 13, 1247-51 (2007) .
- [12] Shrestha, U. K. & Sapkota, S. Etiology and adverse outcome predictors of upper gastrointestinal bleeding in 589 patients in Nepal. *Dig Dis Sci*, 59, 814-22 (2014) .
- [13] Butt, Z., Ali Shah, S. M., Afzal, M., Younis, I., Waqas, M. & Atta, H. Frequency of different types of gastric varices in patients with cirrhosis due to chronic hepatitis C. *J Pak Med Assoc*, 66, 1462-1465 (2016) .
- [14] Kahloon, A., Chalasani, N., Dewitt, J., Liangpunsakul, S., Vinayek, R., Vuppalachhi,

- R., Ghabril, M. & Chiorean, M. Endoscopic therapy with 2-octyl-cyanoacrylate for the treatment of gastric varices. *Dig Dis Sci*, 59, 2178-83 (2014) .
- [15] Monsanto ,P., Almeida, N., Rosa, A., Macoas, F., Lérias, C., Portela, F., Amaro, P., Ferreira, M., Gouveia, H. & Sofia, C. Endoscopic treatment of bleeding gastric varices with histoacryl (N-butyl-2-cyanoacrylate): a South European single center experience .*Indian J Gastroenterol*, 32, 227-31 (2013) .
- [16] Khawaja, A., Sonawalla, A. A., Somani, S. F. & Abid, S. Management of bleeding gastric varices: a single session of histoacryl injection may be sufficient. *Eur J Gastroenterol Hepatol*, 26, 661-7 (2014) .
- [17] Thakeb, F., Salem, S. A., Abdallah, M. & El Batanouny, M. Endoscopic diagnosis of gastric varices. *Endoscopy*, 26, 287-91(1994) .
- [18] Joshi, N. G. & Stanley, A. J. Update on the management of variceal bleeding. *Scott Med J*, 50, 5-10(2005).
- [19] Teng, W., Chen, W. T., Ho, Y. P., Jeng, W. J., Huang, C. H., Chen, Y. C., Lin, S. M., Chiu, C. T., Lin, C. Y. & Sheen, I. S. Predictors of mortality within 6 weeks after treatment of gastric variceal bleeding in cirrhotic patients. *Medicine (Baltimore)*, 93, e321 (2014).
- [20] Franco, M. C., Gomes, G. F., Nakao, F. S., De Paulo, G. A., Ferrari, A. P., JR. & Libera, E. D., JR. Efficacy and safety of endoscopic prophylactic treatment with undiluted cyanoacrylate for gastric varices. *World J Gastrointest Endosc*, 6, 254-9(2014).
- [21] Chiu, P. W. & NG, E. K. Predicting poor outcome from acute upper gastrointestinal hemorrhage. *Gastroenterol Clin North Am*, 38, 215-30 (2009) .
- [22] Bambha, K., Kim, W. R., Pedersen, R., Bida, J. P., Kremers, W. K. & Kamath, P. S. Predictors of early re-bleeding and mortality after acute variceal haemorrhage in patients with cirrhosis. *Gut*, 57, 814-20(2008) .

Citation: Khairy H. Morsy, Asmaa N. Mohammad, Mahmoud K. El-Samman, Walaa A. Ali. *Bleeding Gastric Varices: Frequency and Outcome. ARC Journal of Hepatology and Gastroenterology. 2018; 3(2):1-9.*

Copyright: © 2018 Authors. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.